212 Dizziness

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Definition

"Dizzy" can describe so many different sensations that the clinician's first priority must be to pin down what each patient means by it. The best way to do this is to ask the patient to describe the feeling(s) without using the word "dizzy." Sometimes it becomes apparent that the patient is, in fact, describing fatigue and weakness, visual difficulty, or anxiety, and such situations must be handled as outlined in Chapters 213, 111, and 202. More often, each subjective sensation of dizziness can be identified more precisely as one of four types of dizziness: vertigo, disequilibrium, presyncope, or lightheadedness. The clinical approach to the dizzy patient depends crucially on distinguishing among these various kinds of dizziness, since the differential diagnosis is peculiar to each type.

Vertigo refers to the illusion of environmental motion, classically described as "spinning" or "whirling." The sense of motion is usually rotatory—"like getting off a merry-go-round"—but it may be more linear—"the ground tilts up and down, like being on a boat at sea." Disorientation in space and some sense of illusory motion are the common denominators here. Vertigo always reflects dysfunction at some level of the vestibular system, and these problems are discussed in Chapter 123.

Disequilibrium represents a disturbance in balance or coordination such that confident ambulation is impaired. Symptomatically, some such patients clearly profess that "the problem is in my legs," but others feel "dizzy in the head, too." Common to all patients with disequilibrium is the perception that ambulation either causes the problem or clearly makes it worse. Observation of the patient's gait and a careful neurologic examination are thus essential in evaluating this type of dizziness.

(Pre)syncope means that the patient senses *impending* loss of consciousness. When the patient has, in fact, experienced true syncope (actual loss of consciousness), considerations in Chapter 12 apply. When the patient has not ever actually lost consciousness, the complaint "I feel like I will pass out" should be viewed skeptically, since other types of dizziness may be so described. In such circumstances, the approach to syncope in Chapter 12 may or may not be pertinent.

Lightheadedness is very difficult to describe without using the word "dizzy," but this verbal imprecision is, in fact, very helpful to the clinician. Lightheadedness refers to a sensation "in the head" that is clearly not vertiginous or presyncopal, and that is *not* invariably related to ambulation. This vague "negative definition" emphasizes that the lightheaded patient's description is always hazily imprecise, and even articulate patients are frustrated by the request to describe the feeling without saying "dizzy." Some describe "floating" or feeling "like my head is not attached to my body," being "high," or "giddy." Many will search for a better description but finally concede, "I just feel dizzy, that's all."

Technique

History

The patient's initial spontaneous description is usually the best clue to the type of dizziness. Do not ask leading questions until the patient has attempted to elaborate in his or her own words on "dizzy." As the patient describes symptoms, the clinician must try to interpret those descriptions so as to formulate an initial *hypothesis:* Which of the four types of dizziness does this sound like? When dizziness is episodic or recurrent, ask the patient to recount in detail the setting, circumstances, and events of the most recent or most memorable occurrence(s). Following the patient's untutored tale, analyze the symptoms as you would analyze any symptom: How often does this occur? How long does it last? What seems to you to bring it on? What makes it worse? What makes it better? Is the problem improving or worsening? Are there other symptoms associated with the dizziness?

Often, at the conclusion of the patient's history, a tentative hypothesis has been reached about the type of dizziness, and sometimes even about the specific diagnosis (see below). Table 212.1 illustrates the causes of dizziness among 104 patients studied in a dizziness clinic. Of note is the fact that the most common specific causes of the four general types of dizziness will often be recognizable simply by listening to the patient for a few minutes. For example:

Among patients with vertigo, *benign positional vertigo* is most common. This condition usually afflicts middle-aged and older persons who describe brief episodes of vertigo that are always and only positional, that is, vertigo (and sometimes nausea or vomiting) occurs episodically when the patient moves his or her head into a particular position, as when turning over in bed, looking up, or moving from a standing to supine position. Unlike patients with many other types of vertigo, patients with benign positional vertigo are asymptomatic *except when changing position.* Such a description must be analyzed as outlined in Chapter 123, but the history alone often makes the diagnosis.

Similarly, the most common cause of lightheadedness is hyperventilation, a diagnosis that must be confirmed by testing (see below) but that is usually strongly suspected after analyzing the patient's history. Hyperventilators may initially describe "loss of balance" or "unsteadiness" or "feeling like I will faint," but, in fact, closer questioning usually reveals that this dizziness is very vague, unpredictable, often constantly present but intermittently worse, and inconsistently related to position, activity, or ambulation. Frequently hyperventilators describe associated symptoms that emanate from multiple different organ systems—episodic palpitations, abdominal bloating, paresthesias, and weakness are especially common. This concatenation of seemingly disparate symptoms associated with very vaguely described dizziness is typical of hyperventilation. In fact, the patient

Table 212.1
Final Diagnosis in 104 Dizzy Patients[a]

Vertigo (N = 36)	Lightheadedness (N = 34)	Disequilibrium (N = 19)	Presyncope (N = 3)
Benign positional vertigo (12)	Hyperventilation (23)	Multiple sensory deficits (14)	Orthostatic hypotension (2)
Acute/recurrent vestibular disease (10)	Psychogenic (9)	Frontal lobe apraxia (2)	Micturition syncope (1)
Cerebrovascular ischemia (5)	Anemia (1)	Parkinsonism (1)	
Meniere's disease (4)	Hypothyroidism (1)	Postcataract extraction (1)	
Chronic vestibular disease (3)		Chronic diplopia (1)	
Multiple sclerosis (2)			

[a]No diagnosis was established in 9 patients; 81 patients had a single diagnosis; 12 patients had two simultaneous diagnoses; 2 patients were not "dizzy" at all.

Adapted from Drachman DA, Hart CW. An approach to the dizzy patient. Neurology 1972;22:323–34.

often punctuates the conversation with an occasional deep-sighing respiration of which he or she may be unaware but which is obvious to the alert interviewer.

Multiple sensory deficit syndrome is the most common cause of disequilibrium, and, again, the history alone usually shouts out the diagnosis. These patients are often elderly people afflicted by a variety of ills that summate to impair the patient's ability to ambulate unassisted. Visual impairment, deafness, peripheral neuropathy, painful or disabling orthopedic disorders, and muscle weakness collectively conspire to alter the patient's perception of space, fluidity of motion, and confidence in walking. These people usually complain simply "I am unsteady on my feet" or "I am afraid I will fall" and are dramatically improved by the use of a cane or the supporting arm of a companion. A thorough examination and observation of the patient's gait are essential here, but the history always points the way.

Finally, *orthostatic hypotension* is probably the most common cause of presyncope (these disorders in general are greatly underrepresented in Drachman and Hart's study group in Table 212.1). Such patients feel as if they will faint (not just fall), and this occurs episodically only in the upright position and is invariably alleviated quickly by lying down. The patient often will not have realized the purely "orthostatic" nature of the dizziness, but listening to the patient's description of typical episodes always reveals that crucial connection.

When such "classic" clinical histories are not forthcoming, and especially when the general type of dizziness remains unclear, the clinician can prompt the patient with a variety of analogies of the different types of dizziness. Describing to the patient examples of "dizziness types" often allows the patient to describe vague feelings more clearly. For example, brief vertigo has been experienced by many people after spinning on a revolving stool or chair, in amusement parks, or after overindulging in alcohol. Disequilibrium can be likened to the sensation of walking in complete darkness, especially if drowsy, as may occur when awakening in the night to go to the bathroom. Presyncope is familiar to people who have felt brief orthostatic faintness upon standing up after a prolonged squat, as when gardening or working on the floor. A surprising number of people have, in fact, fainted at some time in the past and can readily identify with the sense of impending faint. Finally, while lightheadedness is difficult to describe, it is almost universally understood in an intuitive, if not experiential, sense. Many lightheaded patients who directly deny the above analogies will immediately brighten when the word is suggested—"Yes, that's it exactly; I feel *light*headed!"—despite the irony that the hallmark of lightheadedness is its *in*exactitude.

Physical Examination

While the history is almost always more revealing than the physical examination of dizzy patients, the examination is important for two reasons.

First, when the history clearly suggests one specific type of dizziness, the physical examination is directed at specific pertinent organ systems. The patient with vertigo must undergo careful neurologic and otologic examination, and the clinical assessment of spontaneous and/or positional nystagmus is essential (see Chapter 128). The patient with presyncope requires careful cardiac and hemodynamic evaluation (see Chapter 12). Disequilibrium demands a thorough general and neurologic examination with specific emphasis on ocular, auditory, and proprioceptive function. The lightheaded patient, so often anxious or depressed, will not and should not be satisfied unless thorough physical assessment excludes the occasional "organic" cause—anemia, thyroid disease, or recurrent cardiac arrhythmias.

Second, the physical examination of the dizzy patient should always include the performance of several dizziness simulation tests. Drachman and Hart demonstrated the usefulness of these tests when they achieved specific diagnoses in 95 of their 104 patients listed in Table 212.1. Any practicing clinician will admire that rate of success. While Drachman and Hart utilized a sophisticated battery of neuro-otologic tests, a few simple "bedside" maneuvers often suffice, as enumerated in Table 212.2.

Table 212.2
Dizziness Simulation Tests

Always helpful
Observe gait, ambulation, turning
Measure blood pressure and pulse with patient supine, sitting, and standing
Observe patient during three minutes of voluntary hyperventilation
Perform Nylen–Bárány maneuvers
Romberg testing (eyes opened and closed)
Occasionally helpful
Valsalva maneuver
Carotid sinus massage
Fistula test
Caloric testing

The patient's blood pressure and pulse should be taken sequentially in the supine, sitting, and standing positions. Orthostatic hypotension is the most common cause of presyncope, and reproduction of the patient's symptoms associated with a fall in blood pressure upon assuming the upright position establishes that diagnosis. Sometimes the fall in blood pressure is delayed, and the patient's blood pressure and pulse should be taken again after a few minutes' standing and/or a brief walk. Intravascular volume depletion and certain drugs are the most common causes of orthostatic hypotension, and usually the pulse rises as the blood pressure falls in such patients. When disease of the autonomic nervous system is the cause, the pulse may not rise when the blood pressure drops.

The patient's gait and Romberg testing should be observed. Disequilibrium disorders are often diagnosed on the basis of these observations (see Chapters 67, 68, and below).

Vestibular function should be tested by examining the patient's extraocular movements for nystagmus and by performing the Nylen-Bárány maneuvers for positional nystagmus and vertigo (see Chapter 127).

Finally, the patient should perform voluntary hyperventilation. Here the patient is placed in the supine position and is asked to breathe deeply and rapidly (about 30 times per minute) through the mouth for as long as 3 minutes continuously. The patient should not be told what to expect. Many normal individuals will develop mild lightheadedness during hyperventilation, and this maneuver is thus useful because it "simulates" lightheadedness—the patient with vertigo, disequilibrium, or presyncope will say, "No, that is not what I feel." Many lightheaded patients, however, are chronic hyperventilators who will very rapidly (within 30 seconds) develop "their symptoms" and experience distress—panic, fluttering eyelids, awkward "thoracic" breathing patterns will emerge, and the patient will spontaneously announce, "That's it! That's my dizziness!" Such a self-realization is an important event among lightheaded patients (see below).

These dizziness simulation tests help in several ways:

1. Often a common specific diagnosis, suspected on the basis of the patient's history, will be confirmed immediately. Positional vertigo, orthostatic hypotension, and hyperventilation can often thus be diagnosed without further ado.

2. These tests can provide experiential examples of certain types of dizziness to allow further refinement of the patient's history when the type of dizziness remains unclear. As noted previously, hyperventilation can simulate lightheadedness, just as Romberg and gait testing can simulate disequilibrium. Occasionally, other tests (Table 212.2) will allow the patient to distinguish among superficially similar sensations. For example, vertigo and disequilibrium are commonly confused, and caloric testing (see Chapter 127) will produce vertigo so that the patient can distinguish the two. (The patient is rarely enthusiastic about this approach.) Lightheadedness and presyncope often overlap subjectively, and the potentiated Valsalva maneuver can mimic presyncope. Here, the patient squats for 30 seconds and then breathes forcefully into a sphygmomanometer tube until a pressure of 40 to 50 mm Hg is reached; the forced expiration is maintained for 10 to 15 seconds. This maneuver decreases cardiac output, simulating impaired cerebral perfusion by diminishing venous return to the heart. The patient will often feel "as if I will faint." This maneuver should not be performed in the elderly patient with cardiac or cerebrovascular disease.

3. A few other tests will (rarely) document a specific diagnosis. As noted above, caloric testing may be useful (see Chapter 127). Carotid sinus massage (see Chapter 12) will occasionally diagnose carotid sinus syncope. The fistula test—precipitating vertigo by introducing air into the patient's ear with a pneumatic otoscope—will help diagnose a perilymphatic fistula.

At the conclusion of the history and examination, the great majority of dizzy patients can be confidently assigned to one of the four categories outlined in Figure 212.1. Further differential diagnosis and treatment then depend on the specific type of dizziness involved. Additional discussion of the approach to vestibular disease and (pre)syncope can be found in Chapters 123 and 127 respectively. A few further points about patients with disequilibrium and lightheadedness are discussed below.

Basic Science

See Syncope (Chapter 12), Motor System and Gait (Chapter 68), Autonomic Nervous System (Section V), and Vertigo (Chapters 123 and 127) for discussion of the mechanisms of dizziness.

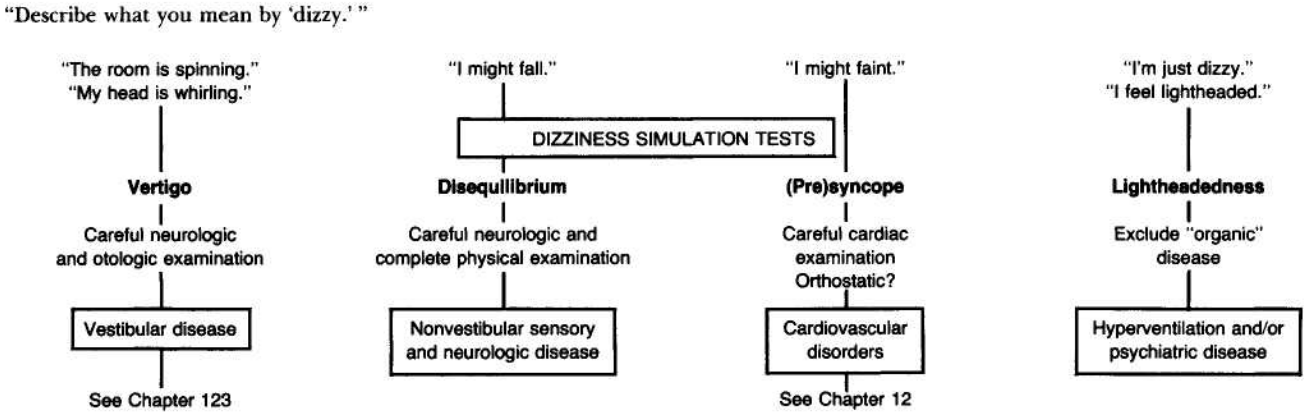

Figure 212.1
Differentiation among the four types of dizziness.

Clinical Significance

Disequilibrium

As noted previously, a detailed general and neurologic examination are essential to the diagnosis of disequilibrium because the syndrome of multiple sensory deficits is so often the cause. The other keys to diagnosis of disequilibrium are:

1. Analysis of the patient's gait.
2. Determining whether the patient's disequilibrium is predictable or episodically unpredictable.

Begin the analysis of gait with the Romberg test. The patient stands upright, feet together, with the arms at the sides. The patient able to maintain balance in this position is asked to close his or her eyes. Loss of equilibrium only with the eyes closed usually suggests disordered proprioceptive and/or vestibular function, while an abnormal Romberg response with the eyes open *or* closed is usually cerebellar in origin. Next, watch the patient walk away from you, turn, and return.

The gait of the patient with *multiple sensory deficits* is usually merely hesitant and apprehensive, and minimal assistance from a cane or companion renders the gait fluid and confident. This type of gait in a patient with two or more major sensory deficits is usually diagnostic. A similar type of gait is seen in some very elderly people who do *not* have multiple sensory deficits. This *"senile gait"* is not associated with senility (dementia), but that unfortunate label remains popular. Such patients' gait, when unassisted, is "scared" and slow; when minimally assisted, it may be brisk and assured.

If the examiner is not observant, he may miss the patient with early *parkinsonism* or *frontal lobe apraxia.* Early in the course of parkinsonism—before the appearance of the classic tremor and "cogwheel" rigidity—disequilibrium and difficulty walking are common findings. At this stage the gait is slow, arm swing is diminished, and turning is disproportionately clumsy. At more advanced stages the parkinsonian gait is "festinating": steps are short and tight, the feet shuffle along, the trunk is bent forward, the arms hang motionless at the sides. Some have likened this appearance to that of the lower body shuffling frantically forward to "catch up" with the rest of the body. Frontal lobe apraxia may be only subtly different in its gait, but thorough neurologic examination usually reveals evidence of dementia and other "frontal lobe" signs in such patients. This gait has been likened to "walking on ice": from a standing position, the patient has difficulty initiating the walk. Steps shuffle to "get going" as if the patient knows how to walk but does not know how to begin. A gentle nudge at the elbow often is the greatest help to these people. Turning is especially instructive as the apraxic patient may appear stuck to the floor, often pivoting around one stationary foot as if one-legged.

Cerebellar ataxia and severe peripheral neuropathy are always easy to recognize. The ataxic patient is not a bit subtle—the patient usually reels unsteadily with short, irregular steps, feet held wide apart as if straddling an invisible barrier, often lurching from side to side or falling, especially when attempting to stop, sit, or turn around. The Romberg test will be abnormal with eyes open or shut.

Peripheral neuropathy is only rarely severe enough itself to cause disequilibrium, but it is a common component of the multiple sensory deficit syndrome, especially in alcoholics or diabetics. The hallmark of the neuropathic gait is that the patient "must see to walk," since proprioceptive function has deserted him. The patient often walks bent forward, watching his feet interact with the ground. This sight can be superficially comical: the legs are usually held apart, each foot is flung out and forward, often higher than necessary, and stomped to the ground as if the patient were wearing frogman's flippers. This gait has been likened to that of a circus clown wearing oversized shoes.

Thus, patients with specific structural neurologic disorders will suffer from "predictable" disequilibrium, that is, their gait will *always* be abnormal when tested. The patient with multiple sensory deficits, parkinsonism, apraxia, or ataxia may have good and bad days, but the gait will never be normal. In contrast, some patients describe disequilibrium that is episodic and unpredictable; examination of such patients' gait may be unremarkable when the patient is asymptomatic. Common causes of such episodic disequilibrium are various vestibular disorders, transient cerebrovascular insufficiency, metabolic disorders, hyperventilation, and psychogenic disequilibrium.

Some patients with *vestibular disease* will neither describe nor admit to vertigo. This is especially true of "central" vestibular disease (see Chapter 123)—acoustic nerve neoplasms, multiple sclerosis, drug (alcohol, anticonvulsants, tranquilizers) toxicity, tumors of the brain stem or cerebellum. But the much more common peripheral vestibular disorders may also cause only a nonspecific sense of disequilibrium, especially in the elderly. Careful neurologic examination is always the clue to the central disorder; the history of disequilibrium induced by positional head changes will usually suggest the atypical presentation of a peripheral disorder.

Transient attacks of *cerebrovascular ischemia* may cause dizziness, and the risk of subsequent catastrophic stroke in such patients always makes the clinician worry about this diagnosis in the elderly or hypertensive patient with episodic disequilibrium. Fisher's classic studies of transient ischemic attacks can help allay or heighten our worry if we remember several of his conclusions:

1. Carotid artery disease rarely causes dizziness or disequilibrium *as an isolated symptom.* In fact, only 8% of 140 patients with *symptomatic* carotid disease complained of dizziness as part of their symptom complex. Thus, for example, the elderly patient with a carotid bruit who describes episodic disequilibrium (in the absence of carotid ischemic symptoms) most likely has two unrelated problems.
2. Disease of the posterior circulation—the posterior cerebral artery and/or the vertebrobasilar system—*does* commonly cause dizziness, usually of the disequilibrium or presyncopal type. However, such dizziness is almost always accompanied by other symptoms—diplopia, vertigo, dysarthria, limb weakness, dysesthesias, ataxia, visual loss—that help incriminate the brainstem or posterior hemispheres. Occasionally, the earliest manifestation of transient ischemic attacks involving the posterior circulation will be episodic dizziness alone, but Fisher found that episodic dizziness that recurs for more than 6 weeks *without* other neurologic manifestations is "never" vascular in origin.
3. Moreover, dizziness accompanied by *only* eighth cranial nerve symptoms (vertigo, tinnitus, deafness) is only rarely vascular in origin. Fisher did describe 10 patients with ischemic symptoms involving only the small internal auditory artery—isolated eighth nerve syndromes can occur in this situation.

4. Finally, positional vertigo or disequilibrium—an extremely common manifestation of peripheral vestibular disease—is "never" caused by cerebrovascular ischemia.

"Metabolic" disorders should be remembered in the patient with disequilibrium. Always ask about all prescription and nonprescription drugs used by the patient. Many people fail to make the connection between the onset of disequilibrium and the use of new (or higher doses of) medication. Hypothyroidism may cause dizziness, ranging from mild episodic disequilibrium to frank ataxia. Episodic hypoglycemia is a fashionable (and usually incorrect) diagnosis among neurotic dizzy patients, but vague dizziness that is always temporally postprandial should raise this legitimate question. Severe anemia may cause vague disequilibrium, as may hyponatremia, adrenal insufficiency, or any cause of hypotension.

Hyperventilation is an extremely common cause of intermittent disequilibrium, even though "lightheadedness" is the usual complaint of hyperventilators. This reminder reemphasizes two important caveats about the patient with disequilibrium or, in fact, any dizzy patient:

1. Because there is overlap among the different subjective sensations of "dizziness," each specific disorder (e.g., hyperventilation) can cause more than one general type of dizziness. Thus, when a patient's disequilibrium cannot be attributed to one of the common causes of disequilibrium described above, it is much more likely that the patient, in fact, suffers from a different "type" dizziness (e.g., vertigo, with its attendant differential diagnosis) than from some obscure cause of "disequilibrium."

2. Thus, our initial hypothesis about the patient's type of dizziness may be incorrect. Sometimes our initial hypothesis must be changed—based on further history, the dizziness simulation tests, findings on physical and neurologic examination, *or because we cannot confirm the hypothesis and must then look elsewhere.* Only this skeptical and circumspect approach will achieve the kind of results reported by Drachman and Hart in Table 212.1.

Lightheadedness

There are four general scenarios that pertain to the patient whose dizziness "sounds" lightheaded:

1. *The patient who is dizzy, i.e., lightheaded, "all the time," constantly.* Such a patient will say, "Yes, I am dizzy right now; it never really goes away." Most of these patients are chronic hyperventilators, and brief hyperventilation during the dizziness simulation tests will exacerbate and/or reproduce all their symptoms. When the examination and simulation tests are all normal, these patients usually suffer from psychogenic lightheadedness, almost always the manifestation of an affective psychiatric disorder. This can be a very difficult problem when the patient insists that the symptoms are due to some feared organic illness, and extensive testing is sometimes required to convince the patient otherwise.

2. *The patient who is lightheaded intermittently and unpredictably.* Most of these patients are also hyperventilators, and the dizziness simulation tests will establish the diagnosis. When voluntary hyperventilation is unrevealing, the young, apparently healthy patient will often have a somatoform or depressive illness; the elderly patient is more often a problem, since masked depression is a common cause, but recurrent cardiac arrhythmias or atypical cerebrovascular symptoms should be excluded.

3. *The patient who is lightheaded in some predictable or reproducible pattern.* Listen carefully to the patient. Never disregard the patient's opinion about what causes the problem, however unlikely this may seem at first glance. Lightheadedness before meals or when hungry may be hypoglycemic. Physical exercise may cause exertional lightheadedness in the patient with severe anemia, aortic stenosis, pulmonary hypertension, pericardial disease, or hypertrophic cardiomyopathy. Positional lightheadedness may be vestibular (head position), related to orthostatic hypotension (body position), or due to carotid sinus stimulation (neck position). The manual laborer, athlete, or artist who becomes lightheaded only when using his right (or left) arm may be looking for disability or sympathy, but he may also be describing the subclavian steal syndrome. The patient who feels lightheaded only during urination may be neurotic, but she may also have micturition (pre)syncope, or even an extra-adrenal pheochromocytoma. When symptoms are reproducible by history, reproduce them on examination; then try to understand the possible mechanism(s).

4. *The patient who is "lightheaded" but, in fact, suffers from a disorder more typically associated with a different type of "dizziness."* Associated symptoms often help most here: concurrent tinnitus or deafness should suggest Ménière's disease or another cochleovestibular disorder; odd behavior, automatisms, retrograde amnesia, or postictal symptoms may point to atypical or temporal lobe epilepsy; palpitations may reflect anxiety, thyroid disease, catecholamine excess, or a primary cardiac disease.

Finally, when you remain confused about the dizzy or lightheaded patient, when nothing "fits," when the examination and dizziness simulation tests are unrevealing, ask a few innocent personal questions: "How's life? Are you happy? Are you under pressure at home or work? Is your family well? Is your marriage good? Do you feel frightened often? Are you depressed? Do you get panicky for no good reason?" Such seemingly ingenuous questions will often "open Pandora's box" in the patient whose dizziness is but one somatic manifestation of either deep personal distress or frank psychiatric illness.

If we look at and listen to the whole person, the examiner need not feel faint just because the patient feels dizzy. Most dizzy patients can be helped, and the clinical process leading to that end can be a provocative and satisfying experience.

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